CHAPTER 8

Psychobiological Perspectives on Attachment

Implications for Health over the Lifespan

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One of the most robust findings to emerge from health psychology over the past 30 years is that individuals in enduring, committed romantic relationships have longer, healthier, and happier lives than unmarried individuals (Kitigawa & Hauser, 1973; Ryff, Singer, Wing, & Love, 2001; Stack & Eshleman, 1998). This effect cannot be attributed to overall social integration, given that individuals' *most intimate* relationships appear to promote health and well-being above and beyond generalized social support (Ryff et al., 2001). Rather, the key variable appears to be the existence of an enduring, emotionally intimate affectional bond (Ross, 1995).

According to attachment theory, feelings of security derived from such bonds play critical roles in regulating our positive and negative emotional responses to internal and external stimuli (Porges, Doussard-Roosevelt, & Maiti, 1994). Because such emotional experiences are directly linked to multiple physiological processes underlying health and disease (Kiecolt Glaser, McGuire, Robles, & Glaser, 2002; Repetti, Taylor, & Seeman, 2002; Ryff et al., 2001), this suggests that attachment relationships—at all stages of life—critically influence physical, as well as psychological, functioning. In this chapter, we review research linking attachment phenomena to specific biological systems and processes that have direct implications for health and well-being over the lifespan.

BOWLBY AND THE PSYCHOBIOLOGY OF ATTACHMENT

Bowlby conceptualized attachment as a fundamentally psychobiological system, especially with regard to its emotion-regulating functions. Specifically, he posited two different "rings" of homeostasis that assist the individual in responding to major and minor stressors (Bowlby, 1973). The inner ring comprises life-maintaining biological systems that govern ongoing physiological adaptation to external demands. The outer ring comprises behavioral (and particularly, interpersonal) strategies for coping and adaptation. From Bowlby's perspective, the integrated functioning of these two levels is critical for optimal self-regulation.

This idea is consistent with extensive research on infants, children, and adults that demonstrates that, in order to understand the mechanisms through which social relationships shape emotional functioning across the lifespan, we must investigate biological, as well as psychological–behavioral, processes of emotion regulation (Repetti et al., 2002; Ryff et al., 2001; Taylor, Dickerson, & Klein, 2002). Such research has taken place across a range of different disciplines, employing divergent methods and aims, and yet the findings are remarkably consistent: Attachment-related processes—specifically, nurturance, caregiving, and support from emotionally primary relationships—fundamentally influence mental and physical functioning over the life course. When viewed collectively, we believe that this body of research indicates two independent (but interacting) pathways through which attachment influences physical health:

1. In infancy, early caregiving experiences establish enduring expectations and orientations regarding attachment figures and also "tune" the brain's sensitivity to stress, predisposing insecurely attached individuals to ineffective physiological and emotional regulation (i.e., ineffective mobilization and management of attentional and metabolic resources in response to environmental demands) and thus poorer long-term health.
2. From childhood to adulthood, attachment-related expectations and experiences shape individuals' cognitive, affective, and behavioral responses to environmental events and their willingness and ability to derive comfort and support from close social ties. As a result, insecurely attached individuals experience higher and more sustained levels of negative affect and lower levels of positive affect, which subsequently influence their long-term health through multiple biobehavioral pathways.

We posit that the joint functioning of these pathways explains the increasing—but as yet undertheorized—body of empirical findings linking attachment insecurity to health-related outcomes such as increased symptom re-

ports and health complaints (reviewed in Feeney, 2000). Both of the aforementioned pathways posit that such links are mediated by emotion-related processes, and thus we begin by outlining the basis for this perspective.

EMOTIONS AS THE LINK BETWEEN ATTACHMENT AND HEALTH

As noted by Mikulincer and Florian (1998), although attachment theory has historically been viewed as a theory of interpersonal functioning, Bowlby (1973) placed considerable emphasis on the role of the attachment system in governing *overall* responses to danger and threat. This is reflected in the increasing attention paid by attachment researchers to the distress-alleviation and emotion-regulation functions of attachment (Feeney, 1995; Mikulincer & Florian, 1998; Mikulincer & Sheffi, 2000; Rholes, Simpson, & Orina, 1999). Specifically, regular contact with a supportive, secure attachment figure is theorized to help individuals sustain positive affect and attenuate negative affect on a day-to-day basis (reviewed in Diamond, 2001).

Individual differences in both infant (Ainsworth, Blehar, Waters, & Wall, 1978) and adult attachment style (Hazan & Shaver, 1987) have also been increasingly conceptualized as indexing different capacities and strategies for emotion regulation (reviewed in Mikulincer, Shaver, & Pereg, 2003). Briefly, infants who did not receive adequate "external" emotion regulation from their caregivers are thought to sustain developmental deficits in their "internal" self-regulatory capacities (see Glaser, 2000) and, consequently, to come to rely on secondary—and suboptimal—emotion-regulation strategies. Specifically, individuals with high attachment *anxiety* tend to maximize experiences of negative affect and to be hypervigilant to threat cues, whereas those with high attachment *avoidance* tend to minimize experiences of negative affect and to direct attention away from threat cues (Mikulincer et al., 2003). Both types of attachment insecurity are also thought to involve the inability and/or unwillingness to derive emotion-regulating benefits from contact with attachment figures (Feeney, 1999).

We address the specific consequences of these strategies in greater detail later. For now, we simply emphasize that adaptive emotional functioning lies at the heart of attachment theory's model of human health and development. Extensive research over the past 30 years has confirmed Bowlby's views in this regard. As researchers have increasingly investigated how and why social and environmental factors influence immediate and long-term health, they have collectively come to emphasize the beneficial effects of positive affectivity and the detrimental effects of negative affectivity for multiple physiological processes, particularly neuroendo-

crine, autonomic, and immune functioning. Because several detailed reviews of this literature are already available (Kiecolt Glaser et al., 2002; Repetti et al., 2002; Ryff & Singer, 2001; Taylor et al., 2002; Taylor, Repetti, & Seeman, 1997), we do not attempt to reiterate these findings. Rather, we highlight the specific role of attachment relationships and dynamics for health–emotion links.

Quite simply, not all emotional experiences are created equal. Multiple research reviews attest to the fact that the emotional context of one's most *intimate* and *important* relationships—parental ties in childhood and romantic ties in adulthood—has the greatest impact on both mental and physical well-being (Reis, 2001; Repetti et al., 2002; Ryff et al., 2001). This is not only because attachment relationships often precipitate some of our most intense positive and negative emotions, but also because the security we (ideally) derive from well-functioning attachments provides an organizing meta-emotional framework for the experience, interpretation, expression, and modulation of positive and negative emotions over the lifespan. Thus, although studies investigating interconnections among relationships, emotions, and health are not typically grounded in an attachment-theoretical framework, we believe that such a framework provides the most powerful and comprehensive unifying explanation for the overall pattern of "cradle-to-grave" associations among these domains.

Before we move on, it is important to note that most of the research we review focuses on negative rather than positive emotions, and particularly on psychological stress. This is not without cause, given that chronic experiences of stress, anxiety, and depression have particularly deleterious physical and mental health consequences (reviewed in Kiecolt Glaser et al., 2002; Repetti et al., 2002). However, research has increasingly focused on the important and independent effects of *positive* emotions on physical and mental functioning (Taylor et al., 2002). For example, positive emotions have been theorized to broaden individuals' thought–action repertoires and build their intrapsychic and interpersonal resources (Fredrickson, 2001), partly through facilitating creative and flexible cognition and adaptive problem solving (reviewed in Isen, 2003). Such conceptualizations have not yet been systematically integrated into attachment-theoretical perspectives on emotion regulation (with some exceptions, such as Mikulincer et al., 2003); clearly, this is a critical priority for future research.

PATHWAY 1: EARLY ATTACHMENT RELATIONSHIPS "TUNE" STRESS-REGULATORY SYSTEMS

Bowlby's lifelong inquiry into the nature of infant–caregiver bonds was prompted by his observation that orphans *deprived* of such bonds during their earliest years developed stark psychosocial deficits. Voluminous re-

search on both animals and humans has since demonstrated that these deficits are linked to alterations in multiple neurobiological processes that appear to be "tuned" in the early years of life by normative maternal care (see Glaser, 2000; Repetti et al., 2002; Schore, 1996; Taylor et al., 2002). To briefly summarize, early infant–caregiver interactions provide for effective and reliable activation and deactivation of stress-regulatory systems in the orbitofrontal cortex that provide the foundation for effective emotion regulation. Understanding these early-developing stress-regulatory systems helps to clarify how and why *adults'* attachment experiences and histories shape their long-term health status. We focus here on those systems for which there is the greatest evidence regarding the impact of early attachment experiences.

Corticotropin-Releasing Factor

The synthesis and release of corticotropin-releasing factor (CRF) from the hypothalamus plays a critical role in mediating behavioral, emotional, autonomic, and endocrine responses to stress, and thus central CRF systems have been extensively investigated as sites for the development and expression of individual differences in stress reactivity (Francis, Caldji, Champagne, Plotsky, & Meaney, 1999; Meaney, 2001). To briefly summarize, environmental demands are processed in the central nervous system by neocortical and limbic centers, and in response the hypothalamus releases CRF and vasopressin into the anterior pituitary, stimulating synthesis and release of adrenocorticotropin (ACTH). This triggers the immediate release of catecholamines (epinephrine and norepinephrine) and subsequent release of adrenal glucocorticoids (most notably, cortisol). The catecholamines and glucocorticoids operate in concert to increase blood glucose levels and influence the specific type, magnitude, and duration of immunological response to environmental demands.

Importantly, increasing levels of glucocorticoids eventually feed back to inhibit CRF synthesis and release, helping to down-regulate (i.e., attenuate or shut down) stress-related hypothalamic–pituitary–adrenocortical (HPA) activation once an adequate neuroendocrine response has been mounted. Notably, however, animal research indicates that these feedback mechanisms—as well as initial CRF gene expression and release—are substantially shaped by early social experiences. Specifically, physical handling of rat pups (by humans) is associated with decreased stress-induced CRF activity and *increased* feedback sensitivity to glucocorticoids, which may account for handled rats' reduced HPA reactivity to stress, reduced behavioral reactivity, and reduced fearfulness (reviewed in Meaney, 2001). In contrast, rat pups that are repeatedly deprived of maternal contact show exactly the opposite effects, and these patterns persist into adulthood (Plotsky & Meaney, 1993).

Of course, both maternal deprivation and human handling are nonnormative rearing conditions for rats; yet additional research has found that variations *within the normal range* of rodent caregiver behavior also influence CRF and HPA functioning. Specifically, pups raised by mothers who exhibited low frequencies of licking, grooming, and arched-back nursing developed heightened CRF, HPA, and behavioral activation in response to stress, whereas pups raised by mothers with high frequencies of licking, grooming, and nursing showed the opposite pattern (Francis et al., 1999). Interestingly, some of these effects might be mediated by opioid mechanisms. Animal research has found that endogenous opioid peptides are released in response to social—and especially physical—contact, whereas social isolation is associated with reduced brain opioid levels (reviewed in Nelson & Panksepp, 1998). Some research suggests that brain opioids may down-regulate CRF activity (McCubbin, 1993), with attendant down-regulation of HPA and sympathetic nervous system (SNS) activity, thereby suggesting another mechanism through which early infant–caregiver interactions shape the CRF system and its interrelated stress-regulatory processes.

The Hypothalamic–Pituitary–Adrenocortical Axis

Multiple studies of animals and humans have documented individual differences in HPA reactivity to stress (Kirschbaum et al., 1995; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Suomi, 1991), and these individual differences correspond to behavioral and self-report measures of emotion regulation. For example, individuals with exaggerated HPA reactivity (indexed by heightened and prolonged cortisol levels) show deficient coping strategies and exaggerated experiences of negative affect (reviewed in Scarpa & Raine, 1997; Stansbury & Gunnar, 1994), and those whose HPA reactivity fails to habituate to *repeated* stressor administration are characterized by low self-esteem, high introversion, high neuroticism, and multiple physical complaints (Kirschbaum et al., 1995).

Such individual differences have direct implications for physical and mental health over the lifespan. As reviewed by Sapolsky (1996), the excess cortisol secretion associated with exaggerated HPA reactivity is associated with neural degeneration in the hippocampus. Negative effects of HPA hyperreactivity on hippocampal function have been detected as early as 12 months of age in humans and have direct implications for memory, attention, and cognition (reviewed in Gunnar, 1998). HPA hyperrreactivity is also associated with impaired immune functioning (Coe, Rosenberg, & Levine, 1988; Webster, Elenkov, & Chrousos, 1997), impaired memory and attentional process (Lupien et al., 1994), and increased risks for a variety of pathophysiological processes and outcomes, including cardiovascular disease, diabetes, hypertension, and can-

cer (Brindley & Rolland, 1989; Henry, 1983; Krantz & Manuck, 1984; McEwen & Stellar, 1993; Truhan & Ahmed, 1989).

Interindividual differences in HPA activity are partially heritable (Kirschbaum, Wust, Faig, & Hellhammer, 1992; Wuest, Federenko, Hellhammer, & Kirschbaum, 2000) but are also influenced by early experiences with stress and caregiving (see also the review in Gunnar & Donzella, 2002; Liu et al., 1997). For example, maternal separation in rhesus monkeys leads to HPA hyperreactivity, along with passive and withdrawn behavior (Suomi, 1991). HPA activity in human children and adolescents varies as a function of multiple family factors, but the most important of these appears to be the quality of maternal care (Flinn & England, 1995). Such effects are long lasting: Children who have lost one of their parents show exaggerated HPA stress reactivity as adults (Luecken, 1998).

Correspondingly, *high* levels of physical affection and infant–caregiver warmth during stressful periods is associated with normal HPA activation profiles (Chorpita & Barlow, 1998; Hertsgaard, Gunnar, Erickson, & Nachmias, 1995). Specifically, secure attachment–as measured by the Strange Situation–has been linked to attenuated HPA reactivity in response to environmental challenges (Gunnar, Brodersen, Krueger, & Rigatuso, 1996; Nachmias et al., 1996). Thus studies of the HPA axis provide some of the strongest evidence for the health implications of early infant–caregiver attachment.

Autonomic Nervous System Functioning

The cascade of neuroendocrine responses to stress, described previously, is also responsible for triggering activation of the sympathetic and parasympathetic branches of the autonomic nervous system, producing the increased heart rate, blood pressure, and sweat production that are the classic hallmarks of stress. Importantly, the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS) have antagonistic effects on autonomic functioning, and thus stress responses such as heart rate acceleration can be brought about by activation of the SNS, withdrawal of the PNS, or some combination of the two. The specific balance of SNS and PNS control over cardiovascular functioning varies from situation to situation (Berntson, Cacioppo, & Fieldstone, 1996), as well as from person to person (Cacioppo, Uchino, & Berntson, 1994).

These patterns have important health implications. Cardiovascular responses to stress that are more SNS driven than PNS driven are associated with exaggerated HPA stress reactivity (Cacioppo et al., 1995), hypertension (Grossman, Brinkman, & de Vries, 1992) and other long-term cardiovascular health risks (Kristal-Boneh, Raifel, Froom, & Rivak, 1998) and immune deficits (Irwin, Hauger, & Brown, 1992). PNS-driven stress re-

sponses appear to be more rapid, more flexible, and easier to disengage than SNS-driven responses (Saul, 1990), and thus robust PNS functioning, typically measured and described as "vagal tone," has been viewed as a key substrate for the development of effective emotion regulation (Porges et al., 1994).

This view is supported by research demonstrating that infants with greater vagal tone are better able to sustain attention to stimuli and to avoid distraction (Porges, 1992; Richards & Casey, 1992), whereas infants with low vagal tone show poor emotional control (Fox, 1989; Porges, 1991) and high behavioral inhibition (Snidman, 1989). Studies of older children have found that vagal tone at ages 4–5 predicts effective emotion regulation 3 years later (Gottman, Katz, & Hooven, 1996) and buffers 8- to 12-year-old children from the negative physical health effects associated with high exposure to marital conflict (El Sheikh, Harger, & Whitson, 2001). In adults, greater vagally mediated heart rate variability is associated with more effective emotional and behavioral responses to stress (Fabes & Eisenberg, 1997), whereas lower levels are associated with depression, anger, mental stress, generalized anxiety, and panic anxiety (reviewed in Brosschot & Thayer, 1998; Friedman & Thayer, 1998; Horsten et al., 1999).

Although the origin and lifetime stability of these interindividual differences have not been definitively established, some researchers have suggested that early distress-alleviating interactions between infants and their caregivers may shape autonomic functioning through their influence on the secretion of oxytocin, a neuropeptide hormone that is critically implicated in both attachment processes and the down-regulation of HPA and autonomic nervous system (ANS) stress reactivity (reviewed in Carter, 1998; Knox & Uvnas-Moberg, 1998; Taylor et al., 2002). One might therefore hypothesize that insecurely attached infants experience less oxytocin-mediated distress alleviation and subsequently develop less PNS-driven patterns of stress reactivity. There is mixed support for this possibility from research on infants and adults (reviewed in Diamond & Hicks, 2004; Fox & Card, 1999); clearly, this is an important area for future developmental research.

Summary and Caveats Regarding Pathway 1

The preceding review is certainly not comprehensive—early social experiences also have lasting influences on dopamine (Depue & Collins, 1999), oxytocin and vasopressin (Uvnäs-Moberg, 1998; Young, 2002), serotonin (reviewed in Glaser, 2000; Repetti et al., 2002), and catecholamines (reviewed in Taylor et al., 2002), which space constraints preclude us from covering in depth. Note, too, that not all "early rearing" effects are best

conceptualized as "attachment" effects—rather, the degree to which such influences are *specifically* attributable to attachment-related dynamics (i.e., those revolving around emotional security) awaits further study.

Along the same lines, we do not mean to suggest that *all* insecurely attached infants and adults suffer from biologically based regulatory deficits. Rather, we expect that correspondences between attachment insecurity and biobehavioral dysregulation depend on the *degree* and *timing* of infant–caregiver relational deficits, in combination with the initial, genetically based psychobiological characteristics of the infant. Thus, for example, infants with a basic predisposition for HPA hyperreactivity who *also* experience inconsistent and unresponsive caregiving should be most likely to manifest both attachment insecurity and biobehavioral dysregulation.

Then, of course, there is the question of longitudinal continuity: Might certain childhood and adult experiences "repair" (or worsen) regulatory patterns? With regard to attachment style, the answer appears to be "yes." Longitudinal data demonstrate that life events can precipitate developmental discontinuities in attachment style from infancy to adolescence and adulthood (Lewis, Feiring, & Rosenthal, 2000; Weinfield, Sroufe, & Egeland, 2000), and thus researchers must take account of ongoing, evolving interactions between infant–caregiver "legacies" and later interpersonal experiences in order to appropriately model links between attachment style and health over the life course. With respect to biobehavioral regulatory patterns, there are mixed findings regarding the stability of individual differences in HPA (Lewis & Ramsay, 1995) and PNS functioning (Bornstein & Suess, 2000; Stifter & Jain, 1996) over the first 12–15 months of life, but we know little about longitudinal stability after that point. This is clearly a critical area for future research, particularly with regard to the possibility that positive and negative life experiences have concurrent, parallel influences on both biobehavioral stress-regulatory systems and attachment representations.

PATHWAY 2: ATTACHMENT AND THE PHYSIOLOGICAL EFFECTS OF CHRONIC EMOTIONAL EXPERIENCES

There has been extensive research on the affective, cognitive, and behavioral manifestations of attachment insecurity and their implications for social functioning and mental health (Cooper, Shaver, & Collins, 1998; Mickelson, Kessler, & Shaver, 1997). Yet over the lifespan these manifestations also impair physical health through their influence on many of the same physiological systems implicated in Pathway 1. Here we outline how this occurs by detailing (1) how attachment-related expectations and experiences shape cognitive processes in a manner that predisposes individuals to certain types of chronic emotional experiences and (2) how these

emotional experiences influence multiple endocrinological, autonomic, and immunological processes that directly influence health.

First, however, the relative importance of attachment *histories* versus *current* experiences bears discussion. Interestingly, whereas adult attachment research has historically emphasized the former at the expense of the latter, research on social support and health has historically emphasized the latter at the expense of the former. Of course, the two constructs are fundamentally interrelated (Feeney & Noller, 1990), and researchers have therefore increasingly called for their simultaneous assessment (La Guardia, Ryan, Couchman, & Deci, 2000). Accordingly, one might best interpret the dimensions of "security" versus "insecurity" discussed here as *cumulative* constellations of individuals' specific attachment histories, generalized attachment-related expectations, and current experiences of need fulfillment.

Attachment, Appraisal, and Emotion

Attachment theory maintains that, through repeated, emotionally relevant interactions with their caregivers, individuals develop stable expectations about themselves and others that come to organize the encoding, storage, retrieval, and manipulation of affect-laden information, particularly information regarding interpersonal experiences (reviewed in Mikulincer et al., 2003). For example, adults with secure attachment styles (as assessed with self-report measures) make more positive and benign interpretations of others' facial expressions (Magai, Hunziker, Mesias, & Culver, 2000), endorse more positive and less negative interpretations of both hypothetical and actual relationship events (Collins, 1996; Mikulincer & Florian, 1998; Simpson, Ickes, & Grich, 1999), make less hostile attributions of others' motives (Mikulincer, 1998), and make more positive interpretations of others' supportive behavior (Lakey, McCabe, Fisicaro, & Drew, 1996). They also make more positive appraisals of their own coping resources (Berant, Mikulincer, & Florian, 2001).

These patterns of cognitive appraisal subsequently shape individuals' day-to-day patterns of emotional activity and reactivity. For example, securely attached individuals report more frequent and intense positive emotions and less frequent and intense negative emotions than insecurely attached individuals (Feeney, 1995, 1999; Simpson, 1990), both in response to everyday events (Pietromonaco & Feldman-Barrett, 1997; Tidwell, Reis, & Shaver, 1996) and to naturally occurring and laboratory-induced stressors (Magai & Cohen, 1998; Mikulincer, 1998; Rholes et al., 1999).

These patterns of emotional experience not only influence social competence, adjustment, and adult affective disorders (Cooper et al., 1998; Mickelson et al., 1997; Repetti et al., 2002) but also provide the

gateway through which social and environmental experiences "get under our skin" (Seeman, 2001) to shape our physical functioning. As reviewed by Seeman (2001), both interpretations of environmental demands and one's resources (social and nonsocial) for meeting these demands are processed first by the neocortex and then fed to the amygdala and hippocampus, leading to systemwide neuroendocrine activation (LeDoux, 1995). Thus information-processing biases that consistently favor negative and threat-related interpretations of environmental events can consistently overstimulate physiological regulatory systems. Here we discuss several systems for which there is the greatest evidence for the deleterious effects of chronic negative emotionality.

Emotions and Hypothalamic–Pituitary–Adrenocortical Reactivity

As we have reviewed, individual differences exist in HPA reactivity that reflect both genetic and early environmental influences. Yet one's HPA response to a particular stressor is also influenced by a constellation of situational factors, particularly those revolving around experiences of negative affect and the extent to which the stressor is appraised as a threat rather than a challenge (reviewed in Blascovich & Tomaka, 1996). Thus attachment experiences and expectations can directly influence individuals' exposure to the cumulative deleterious physiological effects of sustained HPA hyperreactivity through their influence on such emotions and appraisals.

Evidence in support of this view comes from research demonstrating that adults' HPA activity is negatively associated with overall social support (Seeman et al., 1994; Turner Cobb, Sephton, Koopman, Blake Mortimer, & Spiegel, 2000) and positively associated with chronic experiences of negative affect (reviewed in Scarpa & Raine, 1997). Also, whereas most individuals' HPA reactivity demonstrates habituation on repeated administration of a stressor, individuals who perceive themselves to have inadequate coping resources (Kirschbaum & Hellhammer, 1994) or who continue to appraise a particular stressor as threatening (Stansbury & Gunnar, 1994) will fail to show this habituation response, hence heightening the cumulative toll taken on their stress-regulatory systems.

Correspondingly, studies in which individuals' social support perceptions and experiences were specifically manipulated have detected significant effects on HPA functioning. One study of HIV-positive and HIV-negative men found that plasma cortisol levels decreased significantly among those who participated in a structured social support intervention group compared with a control group of nonparticipants (Goodkin et al., 1998). Another study of HIV-positive men (Cruess, Antoni, Kumar, & Schneiderman, 2000) found that over a 10-week period, those who were

randomly assigned to a stress management program showed reductions in both HPA activity and negative moods. On the basis of such findings, one might hypothesize that secure, supportive attachment relationships can attenuate chronic HPA reactivity—and its attendant health risks—through facilitating positive emotions and buffering against negative appraisals of major and minor stressors.

Emotions and Autonomic Nervous System Reactivity

Extensive research has also demonstrated consistent relations between positive and negative emotions and ANS reactivity. For example, studies comparing experimental inductions of positive and negative affect have found that negative affective states are associated with heightened cardiovascular reactivity (Gendolla & Kruesken, 2001), whereas positive affect is associated with enhanced cardiovascular recovery (Fredrickson, Mancuso, Branigan, & Tugade, 2000). Other studies have focused on the degree to which autonomic stress reactivity is sympathetically versus parasympathetically driven. Negative emotions such as anger, hostility, and anxiety are associated with greater sympathetic and less parasympathetic control over heart rate (Sloan et al., 1994), and individuals with SNS-driven patterns of reactivity describe themselves as being chronically nervous and emotionally reactive and as having difficulty dealing with their feelings. Notably, researchers have found that the negative long-term health implications associated with this constellation of emotional and autonomic functioning are moderated by the degree to which such individuals are also socially isolated (Orth-Gomer & Unden, 1990), suggesting the continuing importance of social "buffering" against stress reactivity throughout life. As a further example of this point, Horsten and colleagues (1999) found that among adult women, reduced parasympathetic control over heart rate was associated with being single, living alone, and having low social support.

Other evidence for links between ANS functioning and chronic emotional states comes from research documenting that structured interventions aimed at altering emotional states or reactivity have corresponding effects on autonomic functioning. Positive therapy outcomes for anxiety have been shown to be associated with corresponding increases in parasympathetic control (Friedman, Thayer, & Borkovec, 1993; Middleton & Ashby, 1995), and other studies have found similar effects as a result of structured relaxation tasks (Sakakibara, Takeuchi, & Hayano, 1994) and cognitive interventions aimed at shifting attention *away* from negative and *toward* positive feeling states (McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). Such findings provide further evidence that attachment experiences can directly shape individuals' health trajectories by modulating their day-to-day experiences of negative and positive affect.

Emotions and Immune Functioning

Finally, there is substantial evidence for effects of chronic negative and positive emotions on immune functioning (reviewed in Cohen & Herbert, 1996; Kiecolt Glaser et al., 2002). Studies have assessed a diverse array of markers of immune function, including total numbers of immune lymphocytes, the ratio of different types of lymphocytes, secretion of proinflammatory cytokines and subsequent inflammatory response, cellular response to inoculations, likelihood and duration of illness in responses to infection exposure, and healing speed for minor, controlled wounds. Notably, effects on one parameter do not necessarily extend to others, and the duration and clinical relevance of immunological changes is often not clear.

Nonetheless, the findings consistently demonstrate that emotional states influence immunological functioning (reviewed in Cohen, Miller, & Rabin, 2001). For example, stress and anxiety have been found to be negatively related—and social support positively related—to medical students' immunological responses to hepatitis B inoculations (Glaser et al., 1992). Notably, certain individuals appear more prone to such immunological changes than others. Individuals with SNS-dominated patterns of stress reactivity (Uchino, 1995) or high basal levels of HPA activity (Petitto et al., 2000) are more likely to show stress-related declines in immune functioning. Chronic levels of and predispositions toward positive and negative affect are both related to antibody responses to immunizations (reviewed in Cohen et al., 2001), and research on clinical populations has found that the intensity of depressive affect appears linearly related to immunological effects (Herbert & Cohen, 1993). Correspondingly, studies involving *changes* in positive and negative affect have documented corresponding immunological changes. A 10-week bereavement support group for HIV-positive and HIV-negative men produced significant increases (assessed at a 6-month follow-up) in several different markers of immune functioning and decreases in numbers of physician visits, compared with control group members (Goodkin et al., 1998), and even laboratory-based inductions of negative and positive affect have been shown to influence immune function (Futterman, Kemeny, Shapiro, & Fahey, 1994).

The specific role of social relationships—and the unique provisions afforded by attachment relationships—holds particular promise for future study. Studies of primates have detected both transient and long-lasting changes in multiple parameters of immune function as a consequence of sustained maternal separation or other atypical rearing conditions in infancy (reviewed in Coe et al., 1988). Given the role of CRF in rearing effects on HPA functioning, it is notable that CRF secretion mediates some stress-induced declines in immune functioning, both through direct influences on proinflammatory immune cells and indirectly through its influ-

ences on HPA (Webster et al., 1997) and SNS activity (Friedman & Irwin, 1995). Finally, one intriguing study of HIV-positive teenagers found that those receiving massage therapy over a 12-week period showed increases in certain parameters of immune functioning, as well as reductions in anxiety and depression, compared with a control group that received muscle relaxation therapy (Diego et al., 2001). Given the aforementioned findings regarding the roles of early maternal contact on the development of infants' stress-regulatory systems, this suggests that the types of interactions and behaviors most commonly observed among individuals' most intimate and important relationships—such as regular and prolonged physical contact—might affect immune functioning not only through their emotion effects but perhaps also as a result of touch-induced secretion of neurochemicals such as endogenous opiods and oxytocin and their antistress effects (reviewed in Knox & Uvnas-Moberg, 1998; Taylor et al., 2000, 2002).

Summary and Caveats Regarding Pathway 2

Clearly, the types of day-to-day positive and negative emotional experiences that are shaped by attachment-related experiences and expectations influence not only our overall happiness and relationship quality but also multiple parameters of physiological functioning. This multiplicity of influences is important. Many researchers have argued that, in the modeling of links among stress, emotion, social relationships, and health over the life course, the chronic and combined activation of multiple stress-regulatory systems is more predictive of long-term health status than hyperactivation of any one system in isolation. The collective, cumulative impact of such repeated stress-related activation on the body has been referred to as "allostatic load" (McEwen & Stellar, 1993; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997) and conceptualized as the accumulated wear and tear on multiple organ systems and tissues that results from the body's ongoing physiological adjustments to environmental demands. Over time, high allostatic load is thought to accelerate the aging process by dysregulating cardiovascular reactivity and recovery, blood pressure regulation, HPA axis functioning, parasympathetic nervous system activation, and serotonergic functioning (reviewed in Ryff et al., 2001).

One of the strengths of this conceptualization is its emphasis on *cumulative* risk factors, which necessitates a life-course approach to researching links between social relationships and health. This is certainly consistent with attachment theory's emphasis on lifetime trajectories of security and insecurity and is empirically supported by Ryff and colleagues' (2001) finding that long-term health status is best predicted by considering individuals' *overall* pathways of positive versus negative relationship experiences and the ways in which they build on—or compensate for—one an-

other over time. Along these lines, one important direction for future research concerns the extent to which particular attachment relationships can single-handedly redirect either negative or positive emotion trajectories, thereby changing an individual's long-term health risks. In other words, just how detrimental is a consistently negative, unsupportive, conflictual marriage for an otherwise securely attached individual who generally adopts positive and instrumental appraisals of major and minor stressors? Conversely, how beneficial is a consistently positive and supportive marriage for an otherwise insecurely attached individual?

As noted earlier, longitudinal research that provides coordinated assessments of biobehavioral regulatory processes, relationship experiences, cognitive appraisal processes, day-to-day emotional experiences, and health status would produce invaluable information on the development and maintenance of interconnections among these domains at different stages of the life course and their long-term health implications. It will be particularly informative to try and pinpoint exactly where individuals' information processing biases come into play and the direct and immediate relevance of such biases for physiological processes. For example, one study found that rumination about an emotional task (but not, notably, about an unemotional task) resulted in heightened blood pressure reactivity, and this effect could be successfully extinguished by introducing manipulations that prevented participants from engaging in rumination (Carels, Blumenthal, & Sherwood, 2000). Of course, attachment phenomena are not the only potential influences on such cognitive–affective processes, and thus, in order to appropriately model the relevance of attachment-specific processes, future research must take a variety of individual-difference dimensions (for example, extraversion, hostility, anxiety) into account.

Finally, as discussed earlier, most research in this area has focused disproportionately on the detrimental impact of negative cognitions and emotional experiences, insufficiently theorizing and investigating the specific cognitive, emotional, behavioral, and physiological benefits associated with positive relational experiences and expectations. Thus attachment researchers should consider embarking on future investigations with an eye toward elucidating the role of emotional security in health maintenance and promotion rather than the role of insecurity in health risk and disease.

CONCLUSION

Reflecting on animal research that documents the coordinated coregulation of biological functions between infants and caregivers, Pipp and Harmon (1987) speculated, "it may be that throughout the lifespan we

are biologically connected to those with whom we have close relationships. . . . [H]omeostatic regulation between members of a dyad is a stable aspect of all intimate relationships throughout the lifespan" (p. 651). This model of enduring psychobiological linkage between intimate social partners has yet to be systematically and specifically validated in humans, but it provides a compelling framework for conceptualizing the multiple associations between attachment phenomena and health-related biological processes outlined herein. Most notably, such a model highlights the fact that interpersonal effects on biological functioning may be for either good or ill—just as positive and supportive relationships may continuously optimize our emotional and biological functioning, so too may negative, hostile, and neglectful bonds have the opposite effect. A laudable goal for future research is to trace these biobehavioral processes over the lifespan across multiple functional domains, helping to map the psychobiological processes through which our most intimate and important affectional ties progressively shape our physical, as well as mental, health over the lifespan.

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